

based on a prespecified mechanistic “dose response” relationship), and the result is based on lack of significance (if our null hypothesis had been that there was no treatment effect in the lowest sextile, not being able to reject the null would probably be a low-power issue [Goldberger et al.’s power calculations are, in fact, based on this null hypothesis]—however, our null hypothesis was that the treatment effect would be the same in all sextiles, and we were able to reject this null hypothesis with an, admittedly marginal, significance of 0.05).

Thus, while we would not disagree with Goldberger and colleagues’ caution “don’t believe it,” we might also caution “don’t disbelieve it.”

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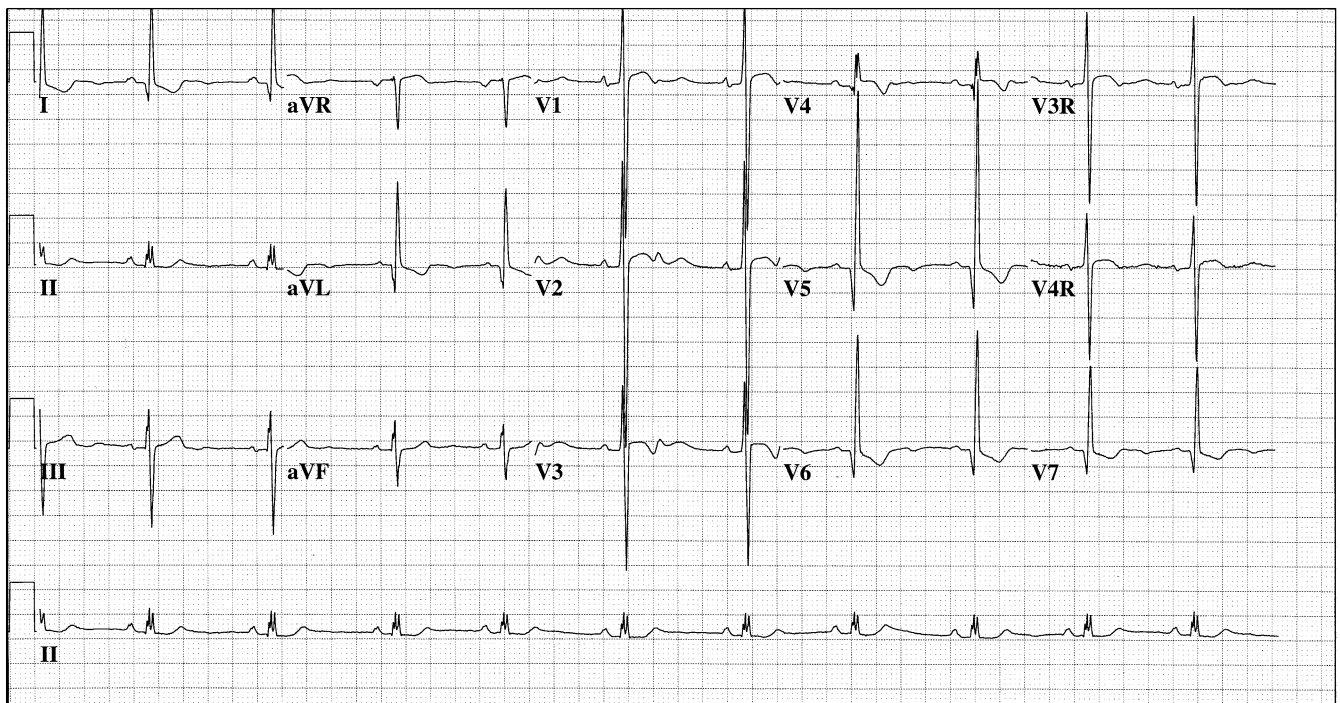
## Congenital Coronary Artery Anomalies: Diagnosable, Premortem?

Dr. Pelliccia’s recent sage editorial comment on congenital coronary artery anomalies (CCAA) (1) establishes an authoritative impetus for the creation of an international registry of youths with CCAA concomitant with a researched analysis of their clinical profile and mode of noninvasive identification. The enormity of this challenge is well appreciated on learning that in 20% of cases wrong sinus coronary malformations are implicated as the cause of

catastrophic unexpected death in young people on the athletic field.

Dr. Pelliccia’s summaries of the admirable presentation by Davis et al. (2), and his citations of the literature review and conclusions of Basso and colleagues (3), instill in the undersigned a warranted enthusiasm and expectation for the noninvasive clinical and electrocardiographic identification of wrong sinus coronary artery anomalies, as has been accomplished for individuals with an anomalous left coronary artery from the pulmonary trunk. Both studies emphasize the importance of a history of exertional chest pain or syncope as a marker of potential sudden death, alerting physicians to the possibility of CCAA in the young athlete. In their report, Basso et al. (3) determined that 37% of the competitive athletes were symptomatic antemortem, and in their literature review of patients with CCAA (among both athletes and nonathletes), Basso et al. found 94% of subjects were symptomatic, and, most importantly, 72% complained of angina, syncope, or dyspnea, on exertion. In this study cohort of young competitive athletes dying suddenly, we are informed that a premortem electrocardiogram (ECG) was normal in all but two of nine patients; however, the configurations of the ventricular ectopy demonstrated by the two Italian professional athletes were unfortunately not clarified (3). In their literature search of the 18 patients under 35 years of age, Basso et al. showed that the ECG was reportedly abnormal in 50% of patients and the stress test positive in 22% (3).

In the four patients identified by Davis et al. (2), two possessed abnormal electrocardiography, one demonstrating ventricular ectopy of right ventricular origin. Sudden death in these patients is presumably due to ventricular tachyarrhythmia, consequent upon vasospasm induced by endothelial injury, or an electrically unstable myocardial cicatrix. The athlete with an anomalous origin of the left coronary artery from the pulmonary trunk is much more readily



**Figure 1.** Anomalous origin of the left coronary artery from the pulmonary trunk. Q-waves and biphasic T-waves in the anterolateral leads I, aVL, V<sub>4</sub>, V<sub>5</sub> and V<sub>6</sub>, suggestive of anterolateral injury in a 14-year-old male Caucasian. Negative U-waves are graphed and there is ST segment elevation in lead V<sub>4</sub>. Kindness of Dr. Gary Webb, Director, Toronto Congenital Cardiac Centre for Adults, Toronto General Hospital.

diagnosable. The presentation is with exertional angina and cardiac palpitations, and the examination discloses cardiomegaly on the basis of dilation of the left ventricle and enlargement of the left atrium. An audible continuous murmur is due to intercoronary collateral flow constituting a left-to-right shunt into the pulmonary artery, and a murmur of papillary muscle dysfunction is present at the apex. The electrocardiographic signature (Fig. 1) is that of a pattern of injury confined to the anterolateral cardiac projection. ST-segment elevation (hypoxemic arteriospasm) and negative U-waves (ischemia) may be graphed. In keeping then with the issues set forth by Dr. Pelliccia, the cardiological mandate is to manufacture a clinical profile and an electrocardiographic marker of the young athlete with CCAA, proposed for the prophylaxis of exertional sudden death.

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